

REMARKS

Claims 1-25 are currently pending in the application. Claims 17-18, 19I-19o and 25 have been withdrawn from consideration due to the Examiner's previous restriction requirement. Claims 5, 11, 16 and 23 have been canceled in the present reply. Claims 1, 7, 8, 12, 13 and 19 have been amended in the present reply. It is submitted that no new matter has been introduced by these amendments, and entry of the same is respectfully requested. These withdrawn, cancelled and amended claims have been withdrawn, canceled or amended without prejudice to, or disclaimer of, the subject matter thereof. Applicants reserve the right to file divisional and continuing applications directed to the subject matter of any claim withdrawn, cancelled or amended for any reason. Applicants do not acquiesce to the propriety of any of the Examiner's prior rejections and does not disclaim any subject matter to which Applicants are entitled. *Cf. Warner Jenkinson Co. v. Hilton-Davis Chem. Co.*, 41 USPQ.2d 1865 (US 1997).

I. Claim Objections**A. Claim 19**

The Examiner objected to claim 19 (I-o) for reciting a non-elected invention. April 17, 2008 Office Action ("OA"), page 3. Applicants respectfully traverse.

Without acquiescing to the propriety of the Examiner's objection, and solely in an effort to expedite prosecution, Applicants have amended claim 19 to remove steps I-o.

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw this pending objection of claim 19.

B. Claims 1, 2, 5, 7, 8, 11-13, 16, 19 and 23

The Examiner objected to claims 1, 2, 5, 7, 8, 11-13, 16, 19 and 23 because these claims recited the acronyms "CREB" and "CRE" which should be spelled out in the independent claims for clarity. OA, page 4. Applicants respectfully traverse.

Without acquiescing to the propriety of the Examiner's objection, and solely in an effort to expedite prosecution,, independent claims 1, 12 and 19 have been amended

to define the acronyms “cAMP” as “cyclic adenosine monophosphate”; “CREB” as “cAMP response element binding protein”; and “CRE” as “cAMP response element.”

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw these pending objections of claims 1, 2, 5, 7, 8, 11-13, 16, 19 and 23.

II. Claim Rejections

A. Claim Rejections under 35 U.S.C. § 112, ¶ 2

Claims 1, 2, 7, 8, 13 and 19 stand rejected under 35 U.S.C. § 112, ¶ 2 as allegedly indefinite for failing to particularly point out and distinctly claim the subject matter the Applicants regard as their invention. OA, page 4. Applicants respectfully traverse.

1. Claims 1 and 19 – Suboptimal Dose

According to the Examiner, claims 1 and 19 are indefinite because “the specification does not provide a standard for ascertaining the appropriate concentration of the CREB stimulating agent to attain a suboptimal dose” and that while “the instant specification describes ‘suboptimal dose’ as that amount yielding a ‘50% or less maximal indicator activity’ that is above natural cellular fluctuations, it is vague and unclear” because “it is not clear as to what ‘cellular fluctuations’ are considered, or what is ‘maximal indicator activity’, etc..” OA, page 4. Applicants respectfully traverse.

The specification provides a number of examples of ways indicator activity can be measured. With these methods, one of ordinary skill in the art can easily determine maximal indicator activity (a ceiling of a particular assay or measure) and can also measure cellular fluctuations (experimental “noise” commonly observed and addressed by routine statistical measures). For example, the specification provides:

[0095] Primary Screen

[0096] A cell-based, high throughput screen (HTS) was designed to assess the effects of compounds on CREB pathway function. Human neuroblastoma cells (SKN-MC) were transfected stably with a reporter construct, in which luciferase was placed under the control of a CRE promoter. With this reporter, increases in CREB pathway function were monitored via fluorescent output. This tester cell line was evaluated fully

using known CREB activators.

[0097] To assess compound activity, the tester line was pre-incubated in the presence of compound and then was stimulated with a suboptimal dose of forskolin prior to lysis. The concentration and time-course for forskolin co-stimulation were calibrated to reproducibly obtain 30% saturation, yielding a 300% range for compound effects. Compounds that produced [greater than or equal to] 100% increase in luciferase signal over control cells stimulated with forskolin alone were progressed to a second round of screening to exclude statistical false positives.

(emphasis added). This passage provides one example of how one of ordinary skill in the art can readily determine a suboptimal dose by appropriately calibrating indicator activity of a cell-based system, such that the meaning of the term “suboptimal dose” is not indefinite under 35 U.S.C. § 112, ¶ 2. The specification also provides that

[0099] Secondary Screens

[0100] Eighty six of the 180 Confirmed Actives were chosen for further analysis based on their structure and potency. To rule out any potential artifactual effects of the reporter gene construct, real time quantitative PCR (QPCR) was used to assess expression levels of known, endogenous CREB-dependent genes in the tester line. Most of the confirmed actives showed comparable potency profiles for both luciferase reporter gene expression and endogenous gene expression.

(emphasis added). This passage addresses the measuring of artifactual effects to control for the presence of natural cellular fluctuations in a cell-based system. It provides additional meaning and context to the term “suboptimal dose” such that one of ordinary skill in the art would understand the meaning of the term within the context of the present invention. The specification goes on to teach another method to measure indicator activity to further one of ordinary skill in the art’s ability to measure and calibrate a suboptimal dose and understand the meaning of the term within the context of the present invention:

[0107] The direct quantitation of CREB messenger RNA (mRNA) following transfection of antisense oligos, was detected using the QuantiGene Expression Kit according to the manufacturer’s protocol (Bayer Corporation; Tarrytown, N.Y.). ... The luminescence of each well was measured in a Wallac Victor² V (Perkin Elmer) plate luminometer at 45°C.

The following passages also provide examples of methods to measure indicator activity and cellular fluctuations such that one of ordinary skill in the art would understand the meaning of the term "suboptimal dose" in carrying out the methods of the present invention:

[0115] Human SK-N-MC cells (see Example 1), stably transfected with a reporter construct consisting of the VIP promoter containing 2 CRE elements together with 2 additional CRE elements from the tyrosine aminotransferase gene driving expression of luciferase, were plated in Iscoves Modified Eagle's Medium (IMEM, Invitrogen, Carlsbad, Calif.), with 2 mM L-glutamine, 0.1 mM Non-essential amino acids, 0.1 mM HEPES, 10% fetal bovine serum at a density of 20,000 cells per well in a 96 well plate format. Cells were maintained at 37° C., 5% CO₂ overnight before transfection with 0.2 µM CREB LNA antisense oligonucleotides. Functional CREB oligos were identified in the bDNA screen described earlier. CREB LNA oligos utilized were: TABLE-US-00003 CREB1: CCTCCgCCgCgTCACTCA (SEQ ID NO.:46) CREB 3: CCACgTAACACACCgCgT (SEQ ID NO.:47) CREB 17: TggCACTgTTACAgTggT (SEQ ID NO.:48)

...

[0117] Cells were transfected with human CREB LNA oligos using Lipofectamine 2000 (Invitrogen, Carlsbad, Calif.) with modifications to the manufacturer's protocol. For each transfection sample, the corresponding CREB LNA oligonucleotide (CREB oligos 1, 3 and 17) was diluted with 25 µl Opti-MEM reduced-serum media (HEPES buffer, 2,400 mg/L sodium bicarbonate, hypoxanthine, thymidine, sodium pyruvate, L-glutamine, trace elements, growth factors, and phenol red reduced to 1.1 mg/L; Invitrogen) to a final transfection concentration of 0.2 µM. The Lipofectamine 2000 was also diluted in 25 µl Opti-MEM reduced-serum media so the ratio of LNA oligonucleotide (µl) to Lipofectamine 2000 (µl) was 1:3, respectively. The diluted LNA oligonucleotide was combined with the diluted Lipofectamine 2000 and incubated for 15 minutes at room temperature for the complexes to form. 50 µl of LNA oligonucleotide-Lipofectamine 2000 complexes were added to each well. The cells were incubated at 37°C. and 5% CO₂ for 16-24 hours. Growth medium was replaced after 5 hours with 100 µl IMEM medium.

[0118] 5 µM HT0712 ((3S, 5S)-5-(3-cyclopentyloxy-4-methoxy-phenyl)-3-(3-ethyl-benzyl)-piperidin-2-one; also known as IPL 455,903)) was added to the [t]ransfected/vehicle treated cells, and incubated at 37°C, 5% CO₂ for 2 hours. HT0712 has the following formula: wherein "Me" means "methyl" and "cPent" means "cyclopentyl". HT0712 can be prepared using the methodology provided in U.S. Pat. No. 6,458,829B1,

the teachings of which are incorporated herein by reference.

[0119] 2 μ M forskolin was then added, and the cells incubated for a further 6 hours, washed briefly with PBS, lysed in the presence of luciferin and luciferase activity measured using a Victor 5 luminometer.

Based on these teachings of the specification, the claims are not indefinite because one of ordinary skill in the art would understand the meaning of “suboptimal dose” within the context of the provided teachings and examples demonstrating various methods to measure indicator activity and cellular fluctuations in order to calibrate a suboptimal dose.

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw the pending rejections of claims 1 and 19 under 35 U.S.C. § 112, ¶ 2.

***2. Claims 2, 8 and 13 – Contacted with Said Test/Candidate Compound
Prior to Contact with Said CREB Function Stimulating Agent***

According to the Examiner, claims 2, 8 and 13 are indefinite because there is insufficient antecedent basis for the limitation contacted with said test/candidate compound prior to contact with said CREB function stimulating agent. OA, pages 4-5. Particularly, the Examiner argues that there is insufficient antecedent basis because “claims 1a, 7g and 12a, recite contacting host cells/cells of neural origin with ‘a test/candidate compound and with a suboptimal dose of a CREB function stimulating agent’.” The Examiner interprets this clause to mean that the test/candidate compound and the CREB function stimulating agent are added together. *Id.* The Examiner states that clarification is required because the specification does not provide a criticality of this limitation. *Id.* Applicants respectfully traverse.

Nonetheless, to address this concern of the Examiner, claims 1a, 7g and 12a have been amended to clarify that the contacting of the host cells with a test compound and with a suboptimal dose of a CREB function stimulating agent can occur simultaneously or sequentially. Thus, in dependent claims 2, 8 and 13, the administration is sequential with test compound contact occurring prior to contact with the CREB function stimulating agent. Support for these amendments is found throughout the specification as filed and particularly at paragraphs [0007], [0008], [0009], [0010], [0013], [0014], [0033], [0034], [00049], [0050], [0065] and [0066].

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw the pending rejections of claims 2, 8 and 13 under 35 U.S.C. § 112, ¶ 2.

3. Claim 7(g) – Cells of Neural Origin are Different From the Host Cells of Step a)

Claim 7(g) recites the limitation “cells of neural origin are different from the host cells of step a)”. According to the Examiner, it is unclear what aspect of the “difference” is referred to because the host cells and the cells of claim 7 are of neural origin. OA, page 5. The Examiner questions whether the “different” limitation refers to “cell type (e.g. from a different nucleus of the brain)” and requests clarification. *Id.* Applicants respectfully traverse.

The specification as filed provides examples of differences between cell types in accordance with the present invention. For example, the specification explains that “in one embodiment, the host cells in the primary screen are neuroblastomas and the cells for the secondary screen are not neuroblastomas.” Paragraph [0053]. The Specification further explains “[p]referably, the host cells in the primary screen are proliferating cells (such as neuroblastoma) and the cells in the secondary screen are nonproliferating, differentiated cells of neural origin (such as neurons (e.g., primary hippocampal cells) and glial cells).” Paragraph [0054]. Applicants assert that these examples render the meaning of claim 7(g) definite as filed.

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw this pending rejection of claim 7 under 35 U.S.C. § 112, ¶ 2.

B. Claim Rejections under 35 U.S.C. § 112, ¶ 1

Claims 1-4, 6-10, 19(a-k), 20-22 and 24 stand rejected under 35 U.S.C. § 112, ¶ 1 as allegedly not enabled by the specification. The Examiner states that the specification is enabling “for a method for identifying a candidate compound for enhancing cyclic AMP response element binding protein (CREB) pathway function by contacting host cells/cells of neural origin with a test compound (e.g. NPY) and forskolin and comparing the indicator activity or CREB dependent gene expression with control cells”. OA, pages 5-6. The Examiner argues, however, that the specification does not enable “the identification of a candidate compound following the same steps by using any CREB function stimulating agent in suboptimal amounts in

combination with any test agent.” OA, page 6. The Examiner concludes that the specification “does not enable any person skilled in the art to which it pertains ... to make and use the invention commensurate in scope with these claims.” *Id.* Applicants respectfully traverse.

When making a rejection on the ground of alleged lack of enablement, the USPTO has the “initial burden of setting forth a reasonable explanation as to why [he/she] believes that the scope of protection provided by [the] claim is not adequately enabled by the description of the invention provided in the specification.” *In re Wright*, 999 F.2d 1557 (Fed. Cir. 1993). Without a reason to doubt the truth of the statements made in the patent application, the application must be considered enabling. *Id.*; see also *In re Marzocchi*, 439 F.2d 220 (C.C.P.A. 1971).

Applicants strongly disagree with the Examiner’s conclusion that the current claim scope is not enabled by the specification as filed. Without acquiescing to the propriety of the Examiner’s rejection, and solely in an effort to expedite prosecution, rejected independent claims 1 and 19 have been amended to recite forskolin as the CREB function stimulating agent.

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw the pending rejections of claims 1-4, 6-10, 19(a-k), 20-22 and 24 under 35 U.S.C. § 112, ¶ 1.

C. Claim Rejections under 35 U.S.C. § 103

To maintain a proper rejection under 35 U.S.C. § 103, the Examiner must meet four conditions to establish a *prima facie* case of obviousness. First, the Examiner must show that the prior art suggested to those of ordinary skill in the art that they should make the claimed composition or device or carry out the claimed process. Second, the Examiner must show that the prior art would have provided one of ordinary skill in the art with a reasonable expectation of success. Both the suggestion and the reasonable expectation of success must be adequately founded in the prior art and not in an applicant’s disclosure. Third, the prior art must teach or suggest all the claim limitations. *In re Vaeck*, 20 U.S.P.Q.2d 1438, 1442 (Fed. Cir. 1991). Fourth, if an obviousness rejection is based on some combination of prior art references, the Examiner must show a suggestion, teaching, or motivation to combine the prior art references (“the TSM test”). *In re Dembiczak*, 50 U.S.P.Q.2d 1614, 1617 (Fed. Cir.

1999). Following *KSR Int'l Co. v. Teleflex, Inc.*, this fourth prong of the prima facie obviousness analysis must not be applied in a rigid or formulaic way such that it becomes inconsistent with the more flexible approach of *Graham v. John Deere*, 383 U.S. 1, 17-18 (1966). 550 U.S. ___ (2007); 127 S. Ct. 1727 (2007). It must still be applied, however, as the TSM test captures the important insight that "a patent composed of several elements is not proved obvious merely by demonstrating that each of its elements was, independently, known in the prior art." *Id.* citing *United States v. Adams*, 383 U.S. 39, 50-52 (1966).

1. Sheriff in View of Herzog – Claims 1, 3-7, 9-12, 14 and 16

Claims 1, 3-7, 9-12, 14 and 16 stand rejected under 35 U.S.C. § 103 as allegedly obvious over Sheriff et al., Reg. Pept. 75-76: 309-318, 1998 ("Sheriff") in view of Herzog et al., PNAS 89: 5794-5798, 1992 ("Herzog"). OA, page 11. According to the Examiner, Sheriff teaches "a method to determine the neuropeptide Y or NPY induced effect on CREB pathway function, such as CREB phosphorylation and CaM kinase activity." OA, page 12. According to the Examiner, Sheriff "further teaches that the treatment of the transfected cells with NPY and forskolin results in a significant increase of luciferase activity as compared to control cells treated with forskolin but not with NPY ..." and that "Sheriff ... demonstrates[s] that the luciferase activity in control cells treated with NPY but not with forskolin is not significantly different from the activity elicited by control cells that are not treated with either forskolin or with NPY (Figure 5, 1st and 2nd bar). OA, page 12. Applicants respectfully traverse.

Applicants respectfully point out that the author of Sheriff does not characterize the activity in control cells treated with NPY but not with forskolin as "not significantly different from the activity elicited by control cells that are not treated with either forskolin or with NPY." Instead, the authors state "[a]ll three effectors, forskolin, thapsigargin and NPY, increased luciferase activity within 4 hr of treatment. ... NPY-induced luciferase activity ranged from 1.8- to 4-fold. The induction of CRE binding and luciferase activity by NPY in SK-N-BE2 cells may involve more than one member of CRE binding transcription factors mediating this effect." Sheriff, page 313, Column 2. Thus, the Examiner's interpretation of these results is contrary to their interpretation as described by the authors, *i.e.*, those of ordinary skill in the art.

Additionally, the work described in Sheriff was directed to elucidating downstream signal transduction pathways beyond the second messenger levels involved in NPY-mediated actions. Sheriff, page 310, Column 1. Sheriff does not teach or suggest screening a plurality of compounds for potential development as candidate cognitive enhancer compounds by determining the ability of said candidate cognitive enhancer compounds to enhance cyclic adenosine monophosphate (cAMP) response element binding protein (CREB) pathway and identifying said test compound as a candidate cognitive enhancer based on the selection parameters of the pending claims. Furthermore, the Examiner acknowledges that Sheriff does not teach repeating the method steps with a range of concentrations of the compound or NPY or teach “the addition of both NPY and forskolin to neuroblastoma cells.” OA, page 12 and 14. The Examiner also acknowledges that Sheriff does not teach contacting cells that are hippocampal neurons. OA, page 15.

The Examiner asserts, however, that “since the disclosure does not specify criticality of the claimed range of doses, optimization within prior art conditions or through routine experimentation is obvious to one skilled in the art.” (OA, page 12 citing MPEP 2144.05) and that “in the absence of unexpected results, it would have been prima facie obvious to one of ordinary skill in the art to combine the teachings of the reference and to treat the cells with both NPY (as the candidate compound) and forskolin” because “[e]ach of these compounds has been taught by the prior art to augment CREB dependent gene expression and increase Y1 mRNA.” Furthermore, the Examiner asserts that Herzog teaches “that the Y1 receptor gene was cloned from a cDNA library derived from the human adult hippocampus.” OA, page 15. The Examiner concludes that “[a]s neuroblastoma, hippocampus comprise cells of neural origin comprising neurons and absent evidence to the contrary, contacting of NPY and forskolin to upregulate Y1 gene expression in *hippocampal* neurons would be obvious....” OA, page 15.

As stated, none of the references cited by the Examiner teach or suggest screening a plurality of test compounds for potential development as candidate cognitive enhancer compounds by determining the ability of said test compounds to enhance the cyclic adenosine monophosphate (cAMP) response element binding protein (CREB) pathway and identifying the test compound as a candidate cognitive enhancer compound based on the selection parameters of the pending claims. Any

attempt to read these limitations into the cited art would constitute nothing more than hindsight reconstruction on the part of the Examiner. *See In re McLaughlin*, 443 F.2d 1392 (C.C.P.A. 1971). Particularly, and as stated, Sheriff is elucidating downstream signal transduction pathways beyond the second messenger levels involved in NPY-mediated actions. Sheriff, page 310, Column 1. The work in Herzog is directed to elucidating receptor subtypes that mediate observed effects of NPY. Herzog, page 5794, Columns 1-2. Neither reference teaches or suggests screening a plurality of compounds to determine the ability of the compounds to enhance the cyclic adenosine monophosphate (cAMP) response element binding protein (CREB) pathway and identifying the compounds as a candidate cognitive enhancer compound based on the selection parameters of the pending claims.

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw the pending rejections of claims 1, 3-7, 9-12, 14 and 16 under 35 U.S.C. § 103 based on Sheriff in view of Herzog.

2. Sheriff in View of Herzog and in further view of Barad – Claims 19(a-k) and 20-24

Claims 19(a-k) and 20-24 stand rejected under 35 U.S.C. § 103 as allegedly obvious over Sheriff in view of Herzog and in further view of Barad et al., PNAS 95: 15020-15025, 1998 (“Barad”). OA, page 16. According to the Examiner, while Sheriff and Herzog “do not teach changes in gene expression after the addition of both forskolin and test/agent compound to the hippocampal neurons...”, Barad

teach[es] a method of elevating cAMP concentrations, using hippocampal slices from mice, that were incubated with increasing range of 4 concentrations of ... rolipram ... in the presence or absence of the adenylyl cyclase activator forskolin The reference also demonstrates that cAMP levels at lower concentrations of rolipram ... were not significantly different from the basal level.... Furthermore, the results demonstrate that the endogenous cAMP levels in cells contacted with rolipram and forskolin are increased relative to the levels in the control group treated with forskolin but not rolipram.

OA, page 17-18. According to the Examiner, based on the foregoing,

it would have been obvious ... to modify the method for elevating Y1 message in neuroblastoma cells as taught by Sheriff ... by using hippocampal neurons. It would, therefore, have also been obvious ... to modify the method for elevating Y1 message in neuroblastoma cells as

taught by Sheriff ... by elevating cAMP levels from hippocampal slices
as taught by Barad...

OA, page 18.

As stated above, neither Sheriff nor Herzog teaches or suggests screening a plurality of compounds to determine the ability of the compounds to enhance the cyclic adenosine monophosphate (cAMP) response element binding protein (CREB) pathway and identifying the compounds as a candidate cognitive enhancer compound based on the selection parameters of the pending claims. Barad does not remedy these deficiencies of Sheriff and Herzog.

Barad “examined the biochemical, physiological, and behavioral effects in mice of partial inhibition of a hippocampal cAMP phosphodiesterase.” Barad, Abstract. The results indicated that “low doses of a type IV-specific PDE inhibitor can act to potentiate and extend LTP at the CA3-CA1 synapse in response to a stimulus that normally induces LTP lasting less than 1.5 hr.” Barad, page 15023, Column 1. The authors interpreted the results as supporting “a correlation between L-LTP and long-term memory” and concluded that “cAMP-driven increases in PKA activity are not only necessary for the transition from short-term to long-term processes ... but can also be sufficient to cause that transition.” *Id.* The authors further concluded that the results of the described studies “indicate that it is possible to increase signaling in the cAMP pathway without significantly affecting basal cAMP concentrations.” Barad, page 15024, Column 2.

Thus, as stated by the authors, Barad studied the effects of partial inhibition of a hippocampal cAMP phosphodiesterase. Barad does not teach or suggest screening a plurality of compounds to determine the ability of the compounds to enhance the cyclic adenosine monophosphate (cAMP) response element binding protein (CREB) pathway and identifying the compounds as a candidate cognitive enhancer compound based on the selection parameters of the pending claims.

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw the pending rejections of claims 19(a-k) and 20-24 under 35 U.S.C. § 103 as allegedly obvious over Sheriff in view of Herzog and in further view of Barad.

3. Sheriff in View of Herzog and in further view of Barad – Claims 2, 8 and 13

Claims 2, 8 and 13 stand rejected under 35 U.S.C. § 103 as allegedly obvious over Sheriff in view of Herzog and in further view of Barad. OA, page 19. According to the Examiner, while Sheriff, Herzog and Barad “do not teach that the cells are contacted with the compound before contacting the CREB stimulating agent” because “the disclosure does not specify the criticality of the claimed sequence ... [i]t would have been obvious to the person of ordinary skill ... to determine the sequential addition of compounds.” OA, page 19. Applicants respectfully traverse.

Without addressing the Examiner's incorrect characterization of what one of ordinary skill would have known, as stated above, neither Sheriff, Herzog nor Barad, singly or in combination, teaches or suggests screening a plurality of compounds to determine the ability of the compounds to enhance the cyclic adenosine monophosphate (cAMP) response element binding protein (CREB) pathway and identifying the compounds as a candidate cognitive enhancer compound based on the selection parameters of the pending claims.

Accordingly, Applicants respectfully request that the Examiner reconsider and withdraw the pending rejections of claims 2, 8 and 13 under 35 U.S.C. § 103 as allegedly obvious over Sheriff in view of Herzog and in further view of Barad.

CONCLUSION

Applicants have properly and fully addressed each of the Examiner's grounds for rejection. Applicants submit that the present application is now in condition for allowance. If the Examiner has any questions or believes further discussion will aid examination and advance prosecution of the application, a telephone call to the undersigned is invited. If there are any additional fees due in connection with the filing of this amendment, please charge the fees to undersigned's Deposit Account No. 50-1067. If any extensions or fees are not accounted for, such extension is requested and the associated fee should be charged to our deposit account

Respectfully submitted,

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/djpelto Reg. No. 33754

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